Functional His-Purkinje system behavior during sudden ventricular rate acceleration in man

MICHAEL H. LEHMANN, M.D., STEPHEN DENKER, M.D., REHAN MAHMUD, M.D., AND MASOOD AKHTAR, M.D.

ABSTRACT During sudden rate acceleration the assessment of human His-Purkinje system (HPS) behavior in the antegrade direction is generally limited by the maximal attainable input frequency due to atrioventricular nodal (AVN) refractoriness. With incremental ventricular pacing, however, faster rates of input into the HPS are achievable. In seven patients with normal HPS function, the HPS response to an abrupt increase in ventricular rate was systematically evaluated with a pacing protocol in which rapid ventricular pacing at any constant cycle length (CL; range 280 to 400 msec) could be initiated at a programmed interval after the last beat of a paced ventricular or atrial basic drive. The following four patterns of HPS behavior accompanying sudden ventricular rate acceleration were observed: (I) 1:1 response without conduction delay, (II) 1:1 response with initial conduction delay but subsequent accommodation, (III) occurrence initially of 2:1 or 3:2 Wenckebach block followed by either subsequent accommodation or repetitive block, and (IV) sustained conduction delay with persistent appearance of the His deflection beyond the ventricular electrogram. The three latter patterns were found to be functional in nature, i.e., dependent on the CL just before rate acceleration, with improved conduction and ability to accommodate facilitated at shorter preceding CLs. Furthermore, with the availability of additional recordings from the right bundle branch, it could be postulated that pattern IV represented retrograde right bundle branch block that was sustained because of repetitive antegrade concealment by impulses conducted retrogradely via the left bundle branch ("linking" phenomenon). We conclude that functional delay and/or block in the HPS is relatively common during constant CL ventricular pacing at rapid rates (i.e., greater than 160/min) and recognition of this fact is important for accurate interpretation of electrophysiologic phenomena.

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DURING incremental atrial or ventricular pacing, both antegrade and retrograde His-Purkinje system (HPS) conduction times are normally constant, even at rapid rates that may be associated with considerable delay in atrioventricular nodal (AVN) conduction.1-4 On the other hand, it is also well established that with an abrupt decrease in cycle length (CL) the relative or effective refractory period (RRP or ERP, respectively) of the HPS may be encountered.5,6 This phenomenon is felt to be responsible for the occurrence of functional bundle branch block7-10 and antegrade 2:1 block in the HPS during rapid atrial pacing.11

Very short constant CLs that may encroach on the RRP-HPS are more readily attained with programmed ventricular stimulation than with atrial pacing since, in the latter case, input into the HPS is limited by AVN refractoriness. The sudden acceleration of rate that occurs at the onset of incremental ventricular pacing, therefore, commonly subjects the HPS to abrupt and potentially marked CL shortening. However, the possible manifestations of such CL changes in relation to retrograde HPS conduction of initial and subsequent ventricular paced beats at the faster rate have not been characterized, perhaps because of the difficulty in differentiating HPS from AVN components of ventriculoatrial (VA) conduction.

We recently studied seven patients with no detectable abnormalities in HPS function who displayed characteristics that facilitated analysis of a variety of retrograde HPS responses to an abrupt acceleration of ventricular rate. A specific pacing protocol was designed that permitted systematic assessment of the effect of the CL before rate acceleration on subsequent HPS response. The observed patterns of HPS behavior are presented and possible underlying electrophysiologic mechanisms and clinical implications are discussed.

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Methods

In order to be included in the present study, patients had to have normal QRS and HV intervals as well as some characteristic that permitted localization (as described below) of site of VA delay and/or block after sudden ventricular rate acceleration. Patients with accessory pathways were excluded. Seven of 231 consecutive patients undergoing complete electrophysiologic studies in this laboratory over a 10 month period satisfied these inclusion criteria: five patients were capable of 1:1 VA conduction at a CL ≤ 300 msee and the two other patients had clear His deflections during incremental ventricular pacing at one or more CLs ≤ 400 msee. The mean age of the three men and four women studied was 47 years (range 14 to 69). Atherosclerotic heart disease was present in four patients, aortic insufficiency in one, and cardiomyopathy in one and one had no structural heart disease. The patients were referred for electrophysiologic evaluation because of ventricular arrhythmias (five), syncope (one), or palpitations (one).

Intracardiac electrophysiologic studies were performed in patients who were in the postabsorptive, nonvesedated state, as previously described.9 The nature of the procedure was explained to all patients and informed consent was obtained. Cardioactive medications were withheld for at least 48 hr before the study. Although complete electrophysiologic data were obtained in each case, only information relevant to this report will be presented. In none of the cases did infranodal block occur during incremental atrial pacing to a CL producing AVN Wenckebach phenomenon. Split His potentials12 were never observed. Furthermore, the occurrence of catheter-induced right bundle branch (RBB) block13, 14 was carefully avoided.

Assessment of HPS response to an abrupt increase in ventricular rate was accomplished with the following pacing protocol. After an 8 beat ventricular or atrial basic drive (S1) of longer CL, a second 8 beat train (S2) of shorter constant CL was initiated at a programmed interval (S1S2) after the last beat of basic drive. All ventricular stimuli were delivered at the right ventricular apex. In the five patients capable of rapid retrograde AVN conduction the basic drive was ventricular and the S1S2 interval was programmed to equal the S2S2 CL. For each of the S2S2 CLs used (ranging in value from 400 to 280 msee), the pacing protocol was repeated with two or three different basic CLs. In the remaining two patients an atrial basic drive (at a CL of 500 or 600 msee) was used with several different programmed S1S2 intervals for each given S2S2 CL. Thus, in all cases it was possible to assess the effect on HPS response of the CL preceding the abbreviated CL of the S2 train.

Results

The various HPS responses observed in these seven patients during rapid ventricular pacing could be categorized into one of four basic patterns. The results will be presented according to this classification.

Pattern I — 1:1 response without conduction delay. This was the most common pattern and was observed in all patients. The shortest S2S2 CL not associated with any detectable HPS delay ranged from 310 to 400 msec (mean 343 ± 34) when the ventricular CL preceding the S2 train ranged from 400 to 700 msec (mean 536 ± 91; figure 1). In these seven cases, therefore, no conduction delays in the HPS were encountered when the first short CL created by the S2 train was 65 ± 8% or more (range 51% to 80%) of the prior CL. Such a CL relationship is consistent with previously reported observations during determinations of HPS refractory periods with single atrial or ventricular extrastimuli.10, 15

Pattern II — 1:1 response with initial conduction delay and subsequent accommodation. During this pattern, despite evidence of slow retrograde HPS conduction after the first short cycle, the VA conduction time (S2A2 interval) quickly returned to a shorter value (figure 2). Such HPS behavior could be clearly documented in the five patients capable of rapid retrograde AVN conduction. Initial prolongation of S2A2 was not associated

![Figure 1](http://circ.ahajournals.org/)

**FIGURE 1.** Pattern I HPS response. Tracings (ECG lead V1, high right atrial (HRA) electrogram, His bundle electrogram (HBE), and time line (T) in A and B are taken from the same patient. S1S1 applies to the basic drive, whereas S2S2 indicates the CL of the subsequent ventricular paced train. Note that there is a 1:1 response with no change in ventriculoatral (SA) conduction time when ventricular CL is abruptly shortened from 600 to 400 msec (A) or from 500 to 350 msec (B). All measurements are in milliseconds. S = stimulus artifact; A = atrial electrogram. The SA interval is measured from S to HRA deflection and listed on top of the HRA electrogram, whereas the S2S2 CLs are indicated at the bottom of each panel. A similar format will be used in subsequent figures.
FIGURE 2. Pattern II HPS response in same patient as in figure 1. After an abrupt decrease in ventricular CL from 600 to 330 msec (A) there is transient prolongation of the SA interval to 165 msec followed by a prompt return (i.e., accommodation) to a value (140 msec) close to the control (135 msec). When the S2S2 CL is further shortened to 310 msec (B) the His deflection emerges with the first beat of the S2 train such that S2H2 = 130 msec (to avoid cluttering, in this and in the two subsequent figures the S2H2 intervals are not labeled) and S2A2 = 185 msec. With the second S2, the His deflection recedes into the ventricular electrogram (where it remains during subsequent beats) and S2A2 shortens to 170 msec. S2A2 decreases to essentially the control value by the next beat and then gradually increases to 150 msec. The latter increase is most likely localized to the AVN. The initial S2A2 and S2H2 delays noted in A and B are not seen in C when an even shorter S1S1 CL (400 msec) precedes the S2 train. In this instance S2A2 increases to a maximum of only 150 msec. Since this is a smaller degree of initial conduction delay than that observed in A and B, despite a shorter S1S1 CL, at least a major portion (if not all) of the initial SA delay in those panels must have been localized to the HPS rather than AVN. V = ventricular electrogram.

with emergence of a retrograde His deflection from the local ventricular electrogram (V2), making it difficult to identify the site of retrograde conduction delay, i.e., AVN vs HPS (figure 2, A). Evidence for conduction delay localized to the HPS, however, was provided by a shorter S2A2 interval during comparable beats when the S1S1 CL was decreased (figure 2, C). Prolongation of S2A2 intervals would have been expected had the AVN been the site of retrograde delay.16

In all patients who exhibited a pattern II response, further shortening of the S2S2 CL produced longer initial S2A2 intervals and concomitant emergence of the His bundle deflection from the V2 electrogram (figure 2, B; table 1). The behavior of S2A2 during subsequent beats again was that of a return towards the shorter values within one or two cycles. This S2A2 shortening during early cycles of the S2 train was followed by a small (5 to 15 msec) increase in S2A2 with subsequent beats. An obvious decrease in S2H2 intervals (HPS accommodation) was associated with S2A2 shortening since the retrograde His deflection was no longer identi

tifiable. The subsequent increase in S2A2 most probably represented AVN conduction delay that can occur at rapid rates, even in the setting of fast retrograde AVN conduction.16 In no case was an increase in S2A2 caused by prolonged stimulus-to-ventricular response latency.

Pattern III — block with or without subsequent accommodation. This pattern was seen in all of the patients who demonstrated pattern II at one or more S1S1 CLs as the S2S2 CLs were further decreased (table 1). The initial delays in the HPS after the first short cycle progressed to a 2:1 or a 3:2 block (figure 3, A and B). Localization of block within the HPS was suggested by the ability of the AVN to conduct in a 1:1 fashion at the same or even shorter S2S2 CL when the S1S1 CL was abbreviated (compare B and C of figure 3). The abolition of conduction delay and block at the same S2S2 CL with shortening of the S1S1 CL (figure 3, C) also strongly supports the functional nature of HPS block during pattern III.

After the blocked beat the S2A2 values returned to
TABLE 1

<table>
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<tr>
<th>Control levels</th>
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<th>Patient 3</th>
<th>Patient 4</th>
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<tr>
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All cycle lengths are expressed in milliseconds.
HE = His emergence (initially) from local ventricular electrogram; SA = subsequent accommodation following blocked beat.

A pattern IV response followed the blocked beat of pattern III.

A occurrence of sustained atrial fibrillation did not permit repeating the pacing protocol with an S2S2 cycle length shorter than 320 msec.

control levels (i.e., S2A2 within 5 msec of S1A1). Subsequent beats were conducted either with less delay followed by accommodation (figure 3, A) or with repeated 2:1 and/or 3:2 block (figure 3, B) for the duration of the S1 train. In one patient a pattern IV response (see below) was observed after the blocked beat.

One can argue that the initial site of retrograde block in A and B of figure 3 may be the AVN, as a result of

FIGURE 3. Pattern III HPS response in same patient as in figures 1 and 2. When the ventricular CL is suddenly decreased from 650 to 320 msec (A) the His deflection emerges from the ventricular electrogram with an S1H2 of 160 msec and S2A2 of 220 msec. These intervals increase further (to 200 msec and 260 msec, respectively) with the second beat of the S1 train. There is complete failure of VA conduction with the third beat. After the blocked beat, S2A2 (140 msec) returns, essentially, to control (135 msec) and with subsequent beats, transient S2A2 prolongation is followed by accommodation as in pattern II (figure 2). Sudden ventricular CL shortening from 600 to 290 msec (B) results in 2:1 VA block without the occurrence of accommodation as observed in A. Repetitive 3:2 and 2:1 block follow. Intact 1:1 VA conduction (C) with only a 15 msec increase in SA at an even shorter S1S1 CL (400 msec) than that used in either A or B, despite short S2S2 CL (290 msec), makes the AVN an unlikely site of block in A and B.
HPS accommodation causing sudden shortening in HH intervals, the latter His deflection being obscured within the V₂ electrogram. This is unlikely because if HPS accommodation had already occurred, HPS delays seen during subsequent cycles (S₂A₂ of 170 and 190 msec in A and B, respectively) would not have occurred. That these delays of 170 and 190 msec can be primarily ascribed to HPS rather than AVN is based on the observation that delays of such magnitude were not seen during subsequent beats, even at shorter S₂S₂ CLs.

Table 1 demonstrates the continuum of HPS patterns II and III as documented in the five patients capable of rapid retrograde AVN conduction. Note how shortening of S₁S₁ CL permits a pattern II response to still be observed at progressively shorter S₂S₂ CLs. In each case, however, pattern III is ultimately encountered as the S₂S₂ CL is further abbreviated (also compare figure 2, B with figure 3, B, both from same patient). HPS accommodation after initial block is also facilitated at shorter S₁S₁ CLs (table 1). Occurrence of 3:2 HPS block during pattern III was noted in association with at least one S₂S₂ CL in all five patients whose data are listed in table 1, although 2:1 block was more frequently observed.

Pattern IV — sustained conduction delay. Pattern IV was observed in three patients (when either an atrial [two] or ventricular [one] basic drive was used). The unique characteristic of this pattern was that the His deflection, once emerging from the local ventricular electrogram with the first short cycle of the S₂ train, remained emerged during subsequent beats (figure 4, A). After initial His emergence, the SH interval either remained constant or shortened over 1 or 2 beats to a steady-state value (figure 4, A). The functional nature of pattern IV behavior is supported by the absence of such marked and sustained retrograde HPS delay when the CL preceding the S₂ train was shortened (figure 4, B).

Discussion

The present report demonstrates that in addition to the expected 1:1 response without conduction delay (pattern I) there are at least three alternative patterns (Nos. II to IV) of HPS behavior that may be observed in man after a sudden acceleration of ventricular rate. Moreover, the various manifestations of conduction delay and block presently described all occurred on a functional rather than pathologic basis in patients having no evident abnormalities of their HPSs. A discussion of the observed phenomena follows.

Accommodation. The rapid accommodation process that occurs during successive beats in pattern II suggests that the HPS is capable of CL-dependent decreases in refractoriness, even when the consecutive impulses propagate during its RRP, at least over a
certain range of S<sub>2</sub>S<sub>2</sub> CLs (table 1). This HPS accommodation phenomenon may be viewed as an extension of the observation that HPS conduction delay encountered by an early ventricular premature beat is decreased with shorter basis CLs. Thus, each short cycle of the S<sub>2</sub> train further shortens HPS refractoriness so that conduction of the subsequent beat is more rapid. Within 1 or 2 beats conduction delay is abolished. An analogous phenomenon has been observed in the antegrade direction during resolution of HPS block induced with rapid atrial pacing (figure 6 in Akhtar et al. 9).

In the variant of pattern II associated with nonemergence of H<sub>2</sub>, the initial site of delay and subsequent accommodation is most probably localized to the RBB. On the other hand, the second variant of pattern II, which is associated with initial emergence of the His deflection, involves a bilateral HPS accommodation process and/or resolution of functional retrograde bundle branch block (see “linking” phenomenon below). This is because it has been demonstrated that, during right ventricular pacing, emergence of the His deflection from the ventricular electrogram after a single short cycle usually indicates retrograde functional RBB delay and/or block, with His activation occurring preferentially via the left bundle branch (LBB). 18

The relatively small number of beats required for HPS accommodation to occur, as described in this report, is in contrast to the several hundred cycles that were necessary, in the experimental study of Janse et al. 19 for attainment of a new steady-state ventricular refractory period after sudden CL shortening. Since canine His-Purkinje tissue displays similar cumulative effects of CL on refractoriness, 20 it is conceivable that, had a longer duration of basic drive (i.e., greater than 8 beats) been used in the present study, more than 1 or 2 beats might have been required to complete HPS accommodation. We used an 8 beat basic drive since that number of S<sub>1</sub> is customarily used during clinical electrophysiologic studies.

Wenkebach block. Eventually, as S<sub>2</sub>S<sub>2</sub> CL is further shortened, even the LBB-His axis may not be able to accommodate and complete bilateral HPS block (pattern III) may result (table 1). As is typical of Wenkebach periodicity, HPS conduction time returns to control after the blocked beat. However, due to slow conduction of the beat preceding the one that blocks, the “effective” CL created by the blocked beat (and registered retrogradely beyond the site[s] of block) may be shorter than twice the S<sub>2</sub>S<sub>2</sub> CL. Moreover, CL dependent shortening of refractoriness might also occur in the tissue proximal to or at the site(s) of block. Due to these factors relative refractoriness may be of shorter duration after subsequent short cycles of the S<sub>2</sub> train than at the onset of rate acceleration. This would result in less HPS conduction delay and permit accommodation to occur. The basis for a greater likelihood, at shorter S<sub>2</sub>S<sub>2</sub> CLs, of HPS accommodation occurring after a blocked beat (table 1) is not clear, but may be related to a higher level of impulse penetration into the region(s) of block when S<sub>2</sub>S<sub>2</sub> CL is shorter.

At even shorter S<sub>2</sub>S<sub>2</sub> CLs, however, accommodation after initial block may no longer be possible, perhaps in part due to excessive prolongation of RRP-HPS (beyond the site[s] of block) after sudden cycle lengthening 21 created by the blocked beat. The repeated occurrence of block (usually in 2:1 fashion) represents the retrograde counterpart of functional antegrade 2:1 HPS block that may be seen during incremental atrial pacing. 11 On the other hand, functional 3:2 block in the HPS, which occurs with either variant of pattern III, has not been previously reported, although occasionally a similar phenomenon may be seen in the antegrade direction. 22 In a recent preliminary report 23 that did mention the occurrence of retrograde HPS Wenckebach block in man, the functional nature of this response was not delineated.

It is well recognized that under abnormal conditions the HPS may display Wenckebach periodicity at both fast and slow rates, as evidenced clinically in patients with conduction system disease 12, 24 and experimentally in chemically 25, 26 or electrically 27 depolarized Purkinje fibers. The present findings clearly demonstrate, however, that an HPS Wenckebach phenomenon may also be manifest on a purely functional basis at rapid rates (i.e., greater than 160/min).

Linking phenomenon. Sustained marked prolongation in retrograde HPS conduction time during constant CL pacing, as is characteristic of pattern IV behavior, has not been previously described. The functional nature of this phenomenon as presently shown (figure 4), argues against the possibility that the occurrence of pattern IV signifies persistent generalized conduction delay in the HPS. For similar reasons, it is unlikely that a fixed impairment in retrograde RBB conduction can be invoked. In this regard, it is important to reemphasize that there was no evidence of traumatic (antegrade) RBB block as a possible explanation for pattern IV HPS behavior (note normal supraventricular beats before and after the S<sub>2</sub> train in figure 4).

A dynamic process, such as depicted schematically in figure 5, may be a more tenable mechanism for the

*Unpublished observations.
occurrence of sustained retrograde HPS conduction delay during rapid ventricular pacing. The first beat of the $S_2$ train (following a presumed atrial drive) conducts normally to the bundle of His via the RBB, since the latter is ipsilateral to the right ventricular site of stimulation (figure 5, A). Emergence of the His deflection with the second $S_2$ beat (i.e., after the first short cycle) typically signifies that retrograde functional block along the RBB-His axis has occurred with concomitant transeptal activation of the left-sided HPS and subsequent impulse propagation to the His bundle via the LBB (figure 5, B). The same impulse also reenters the RBB in antegrade fashion but encounters refractoriness and blocks (figure 5, B). It should be mentioned in this connection that if antegrade conduction in the RBB had been obtained instead of block, true ventricular reexcitation, i.e., so-called macro-reentry within the HPS, would have occurred (figure 6, A).

With the third beat of the $S_2$ train (figure 5, C) retrograde block in the RBB occurs due to antegrade concealment engendered by the previously conducted impulse. Persistence of retrograde block in the RBB, therefore, is “linked” to electrophysiologic sequelae of the previous beat. Despite right-sided retrograde block, the impulse is still able to cross the septum and travel along the LBB, which may have accommodated (figure 5, C) as a result of the short preceding CL. Propagation along this route, therefore, may be faster and the SH interval shorter than was the case with the previous beat. However, the new impulse also antegrade reenters the RBB and blocks (figure 5, C), leaving in its wake refractoriness to be encountered, in turn, by the next paced beat. Thus, the linking phenomenon tends to perpetuate itself.

More complete accommodation of the LBB-His axis occurring with an additional short cycle (figure 5, D) might account for the further shortening of the SH interval sometimes observed during pattern IV (figure 4, A and figure 6, B). The linking will persist, however, unless the site of retrograde RBB block is able to progressively advance more proximally (perhaps due to “local” HPS accommodation) (figure 5, C through E) so that ultimately retrograde His activation via the RBB can occur (figure 5, F). Conduction might also be improved at the site of block if a summation phenomenon at the site of retrogradely and antegrade blocked impulses occurs and somehow produces a salutary effect on retrograde propagation of the subsequent paced beat. Although the exact mechanism cannot be determined from this study, the occurrence during pattern IV of proximal migration of retrograde block site is strongly suggested by the shortening of the H-RB interval with eventual disappearance of the RB deflection observed in one patient (figure 6, B). An analogous phenomenon, i.e., farther migration of block site with progressive improvement in conduction along the His-RBB axis, has been documented during repetitive antegrade functional RBB block as well.

**Clinical implications.** The different types of HPS behavior described in this report would be expected to occur during rapid ventricular pacing (where “$S_2S_3$” is obviously random), ventricular tachycardia, and so-called antidromic reentrant tachycardia in patients with antegrade functioning accessory pathways. In such settings, therefore, the possibility of functional HPS delay and/or block should be considered as a possible explanation for His activation patterns (especially patterns III and IV) that might otherwise be ascribed to HPS pathology or drug effect. Furthermore, occur-
FIGURE 6. Elucidation of pattern IV HPS response with RBB recording. Tracings from another patient. Format similar to that of figure 4, with addition of RB recording. A and B, S1 drive originates from HRA site (S1s1 CL = 600 msec) and S2 is of ventricular origin (S2s2 CL = 300 msec). When S1s2 = 800 msec, with corresponding V1s2 of 600 msec (A), there is retrograde HPS delay (S2H2 = 170 msec) encountered by the second S2 beat followed by right ventricular reexcitation due to bundle branch reentry. Note that the H-RB interval during reentrant beat is shorter than during sinus (40 msec), a finding compatible with HB and RB activation via the left bundle. The macroreentrant beat renders the ventricle refractory to the third S2, but normal retrograde conduction occurs with the next beat. This is followed, however, by pattern IV HPS behavior with a constant S2s2H2 of 130 msec. Since the RB deflection is clearly visible preceding the bundle branch reentrant beat, absence of a RB deflection during the presumed linking phenomenon may indicate a site of retrograde block and consequent concealment along the RB-His axis between the recording sites of the His bundle and RBB. When S1s2 is shortened to 700 msec, with corresponding decrease in V1s2 to 500 msec (B), there is less retrograde HPS conduction delay (S2H2 = 145 msec) occurring with the second S2 beat than is the case in A. Despite antegrade RB activation in B, the shorter S2s2RBH2 (170 msec) compared with the corresponding beat of A falls within the effective refractory period of the distal RBB and does not permit ventricular reexcitation. Instead, the linking phenomenon is observed for the duration of the S2 train. Note progressive S2s2H2 shortening, i.e., accommodation to 130 msec, a value identical to that observed in A. Disappearance of the RB deflection after the fifth beat may reflect migration of site of retrograde block and concealment along the RB-His axis from a location distal to the RB recording site to an area between the His and RBB recording positions. Such migration would be a prerequisite for abolition of the linking phenomenon. In addition, note change in H2s2RB2 interval in second through fifth S2 beats (25, 30, 15, and 15 msec, respectively), indicating initial delay along the retrograde-left to antegrade-right bundle pathway with subsequent retrograde activation of the RB recording site via the RBB. This occurs despite continued retrograde His activation via the LBB. Such altered local activation is also consistent with migration of RBB block site. C. Morphology of and relationship between the His bundle and RB deflections as observed during retrograde refractory period studies (with S1s2 CL of 600 msec and S2s2 CL of 300 msec). Note that H2s2RB2 is 25 msec, a value identical to that seen in A and B.

rence of HPS block (i.e., pattern III) may be responsible for the initiation of orthodromic reentrant tachycardia during rapid ventricular pacing in some patients with Wolff-Parkinson-White syndrome.31

It should also be appreciated that persistent V His prolongation with antegrade RB activation occurring in association with sustained bundle branch reentry may be simulated by a linking phenomenon during ventricular tachycardia, in which case the HPS is responding, rather than contributing, to the accelerated ventricular rate. Differentiating these two entities may require careful analysis of mode of initiation of the tachycardia as well as a correlation of spontaneous changes in the VH interval with alterations in CL.

A linking-type mechanism has also been suggested as an explanation for sustained functional antegrade bundle branch block.7,33 Thus, the linking phenomenon together with a mechanism for its potential dissolution (i.e., migration of block site) provide a unifying concept for understanding occurrence and possible resolution of both antegrade and retrograde sustained functional bundle branch conduction delay or block.

Finally, it should be pointed out that the patterns presently described may not constitute the entire spectrum of HPS behavior during a sudden increase in ventricular rate, particularly in patients with abnormal HPS. Moreover, the full range of S1s2 CLs (for given S1s2 CLs) normally associated with each pattern has yet to be determined.

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